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Four weeks of high cadence training alter brain cortical activity in cyclists

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ABSTRACT

Exercise at different cadences might serve as potential stimulus for functional adaptations of the brain, because cortical activation is sensitive to frequency of movement. Therefore, we investigated the effects of high (HCT) and low cadence training (LCT) on brain cortical activity during exercise as well as endurance performance.

Cyclists were randomly assigned to low and high cadence training. Over the 4-week training period, participants performed 4 h of basic endurance training as well as four additional cadence-specific exercise sessions, 60 min weekly. At baseline and after 4 weeks, participants completed an incremental exercise test with spirometry and exercise at constant load with registration of electroencephalogram (EEG).

Compared with LCT, a greater increase of frontal alpha/beta ratio was confirmed in HCT. This was based on a lower level of beta activity during exercise. Both groups showed similar improvements in maximal oxygen consumption and power at the individual anaerobic threshold.

Whereas HCT and LCT elicit similar benefits on aerobic performance, cycling at high pedalling frequencies enables participants to perform an exercise bout with less cortical activation.

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KEYWORDS
Alpha/beta ratio; brain function; EEG; VO_{2MAX}

1. Introduction

Exercise is known to elicit adaptations in several organ systems, which inter alia lead to metabolic, cardiovascular and cardiorespiratory changes (Kenney, Wilmore, & Costill, 2015). As the activation of motor units and thereby the movement itself presupposes signalling from the central nervous system, brain function is undoubtedly linked to these processes. Consequently, understanding brain function during exercise has become a major interest in exercise science (Crabbe & Dishman, 2004). Different techniques have been used to investigate perceptual and affective responses to exercise. Especially, the electroencephalogram (EEG) serves as a practical method to assess brain cortical activity during motion as it does not require the restriction of movements (Reis, Hebenstreit, Gabsteiger, Von Tscharner, & Lochmann, 2014). Therefore, knowledge on the dose–response relationship of acute exercise and brain cortical activity has predominantly been generated by investigating EEG rhythms (Hottenrott, Taubert, & Gronwald, 2013).

Previous studies have used frontal alpha/beta ratio to assess the athlete's level of arousal, because a decrease in beta activity and an increase in alpha activity have been associated with decreased arousal or a decline in vigilance (Nielsen, Hyldig, Bidstrup, González-Alonso, & Christoffersen, 2001; Nielsen & Nybo, 2003). In line with these findings, high alpha activity and/or low beta activity in the frontal lobe were observed after running at high intensity (Brümmer, Schneider, Abel, Vogt, & Strüder, 2011; Schneider et al., 2009). Moreover, an increase of frontal alpha/beta ratio was found during exercise in the heat as well as in normothermic conditions (room temperature: 20°C) (Ftaiti, Kacem, Jaidane, Tabka, & Dogui, 2010; Kacem et al., 2014). This change has been related to an increase in rating of perceived exertion (Nybo & Nielsen, 2001) and reduced time to exhaustion during stationary cycling (Ftaiti et al., 2010). An increase of frontal alpha/beta ratio during exercise is also suggested by the transient hypofrontality hypothesis (Dietrich, 2006), which posits that intense exercise causes a deactivation of the frontal brain region due to limited resources of the brain. So far, changes in frontal alpha and beta activity during exercise have only been investigated in response to acute exercise bouts (Ludyga, Gronwald, & Hottenrott, 2015c). Hence, it remains unclear whether or not exercise training may alter this functional response of the brain.

For the provocation of long-term functional adaptations to exercise, scientists usually follow the overload principle in the quantification of loads (McArdle, Katch, & Katch, 2010). Applying this principle to elicit changes in brain function, an exercise intervention must provide sufficient stimuli to the central nervous system. Hottenrott et al. (2013) have reported that EEG spectral power is modulated by movement frequency. In this respect, an increase of pedalling frequency from 90 to 120 rpm led to higher alpha and beta activity, whereas a decrease to 60 rpm reduced activity in both frequency bands. The effect of cycling cadence on brain cortical activity was also observed earlier by Schumann and Seibt

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(1993). Based on those findings, we attributed high potential for altering brain cortical activity to cadence-specific training.

In our present study, we investigate the effects of HCT and LCT on brain cortical activity during exercise and aerobic performance. Based on the relation between pedalling frequency and EEG activity (Hottenrott et al., 2013; Schumann & Seibt, 1993), we expected changes in frontal alpha/beta ratio during exercise to occur after HCT only. As a lower level of arousal has been confirmed during an exercise bout of submaximal intensity in cyclists with higher aerobic fitness compared to peers with lower aerobic fitness (Ludyga, Gronwald, & Hottenrott, 2015b), a lowering of the alpha/beta ratio with increased aerobic power was expected.

2. Methods

Participants

Fifteen male and seven female cyclists (average values for age: 27 ± 4years; body mass: 73.5 ± 8.2 kg; height: 178.0 ± 6.9 cm; VO_{2MAX}: 52.6 ± 3.7 (m)/45.9 ± 4.8 (f) ml · min^{-1} · kg^{-1}) were directly recruited from local sports clubs to take part in an experiment. Eligible participants had to meet the following inclusion criteria: (1) non-smokers, (2) a weekly training volume of 8 h (at least 5 h cycling) and (3) no health problems. A preliminary screening process including personal history, electrocardiogram at rest, orthopaedic check-up and physical activity questionnaire (PAR-Q) was employed to ensure a low risk of complications during exercise. Before the study commenced, participants received information on possible risks and benefits associated with exercise testing and were familiarised with the laboratory setting. Afterwards they provided informed written consent. All procedures were in line with the Declaration of Helsinki and ethical approval was granted by the local ethics committee.

Design

The experimental protocol has been described in Ludyga, Hottenrott, and Gronwald (2015a) and consisted of (1) baseline testing, (2) a 4-week training period and (3) post-testing procedures. For the exercise intervention, study participants were randomly assigned to either a high (HCT; n = 8 m/3 f) or low cadence training (LCT; n = 7 m/4 f). At baseline and after the training period, participants' aerobic power and brain cortical activity during exercise was assessed in two separate laboratory visits. On the first session, they completed an incremental test on a cycling ergometer. After 4 days, participants' brain cortical activity was recorded with EEG during cycling at constant load. For post-testing, these procedures were repeated after the training intervention.

Exercise testing

After assessing body mass (Tanita, BC-545 Inner Scan, Germany), participants performed an incremental test with spirometry (Cortex Medical, Metamax 3b, Germany) on a cycling ergometer (FES, Germany) at a pedalling frequency of 85 to 95 rpm. From initial 100 W (m)/80 W (f) load was increased by 25 W/3 min. The test was continued until participants reached volitional exhaustion, which was defined as inability to maintain a cadence of at least 75 rpm. After each stage, blood lactate concentration was analysed with the enzymatic–amperometric method (Super GL Ambulance, Dr Mueller Geraetebau, Germany) in 10 ul capillary blood taken from an ear lobe. Heart rate and respiratory data were continuously recorded. Following the processing of collected data with WinLactat 3.1 (Mesics, Germany), the individual anaerobic threshold was defined as the lowest quotient of lactate·$power^{-1}$ + 1.5 mmol · l^{-1} (Dickhuth et al., 1991).

On a separate laboratory visit, participants' brain cortical activity was assessed during cycling. Prior to exercise testing, resting EEG was recorded over 90 s with eyes closed followed by 90 s with eyes open while sitting on the ergometer. Subsequently, study participants completed a warm-up (WU) (7 min at 100 W[m]/80 W [f]), a 30-min exercise bout at constant load (100% of the individual anaerobic threshold) followed by a cool-down (CD) (3 min at 100 W [m]/80 W [f]). Brain cortical activity was recorded while cyclists maintained a cadence of 90 rpm. During the test, heart rate, time and cadence were displayed on a monitor, which was placed in the participants' natural viewing direction. Prior to testing, participants were instructed to maintain a stable body position and avoid head movements throughout the cycling exercise.

Standardisation procedures included the use of 24 h recall protocols to match participants' nutrition at each laboratory visit. They were instructed to have their final meal 90 min before testing and refrain from consuming caffeine in any form and dosage. The nutrition protocols were controlled for food and caffeine intake. Furthermore, baseline and final testing took place at the same time and day of the week. All laboratory tests were performed at an environmental temperature of 20°C.

EEG recordings

The EEG application process followed the guidelines of the American Clinical Neurophysiological Society. Prior to exercise testing, a flexible, breathable EEG cap (ActiCap) was mounted on the participants scalp. Electrodes were placed over the frontal brain region (Fz, F3, F4, F7 and F8) and filled with SuperVisc electrode gel (EasyCap GmbH, Germany) to reduce impedances below 10 KΩ. FCz was used as reference and AFz as ground. The arrangement of the electrodes followed the international 10:20 system (Jasper, 1958). Active Ag/AgCl electrodes were used as they are known to register electro-cortical activity without movement artefacts even during intense exercise (Brümmer, Schneider, Strüder, & Askew, 2011). The EEG signal was amplified with the QuickAmp system (BrainVision, Germany) and recorded at 512 Hz with vision Recorder (BrainVision, Germany). Collected EEG data was then processed with BrainVision Analyzer 2.0 (Germany).

Following a reduction of the sampling rate to 256 Hz, high and low-pass filters were applied, so that the frequency range from 3.0 to 40 Hz was used for analysis (time constant 0.0318 s; 24 dB · $octave^{-1}$). In the next step, five 60-s segments were extracted from continuous EEG recordings. The segments represented the 5th min of the warm-up, the 10th

(M1), 20th (M2) and 30th (M3) min during exercise at P_{IANS} as well as the 3rd min of the cool-down. A systematic protocol based on a standard threshold method was applied to accurately tag artefacts. In a subsequent manual inspection, those artefacts were either confirmed or rejected. Within the remaining artefact-free epochs, five consecutive 2-s segments were selected for further analysis. A Hanning window (20%) was applied to those segments, and thereafter, FFT was used to calculate mean power spectra in the alpha (7.5–12.49 Hz) and beta frequency range (12.5–32 Hz). For both bandwidths, mean activity was exported and normalised to the average power of the same frequency band during the recordings at rest (eyes open). Similar to previous studies (Ftaiti et al., 2010; Nybo & Nielsen, 2001), the relative alpha/beta ratio was calculated from normalised mean activity to reduce interpersonal and trial-to-trial variation. The EEG data acquisition and processing methods used in the present study have been shown to provide acceptable levels of test–retest reliability (ICC = 0.7–0.8) for the alpha and beta power during cycling at 90 rpm (Ludyga, 2014).

Exercise training

HCT and LCT engaged in 4-h unsupervised endurance training weekly, whereby individual heart rate targets (70–80% P_{IANS}) were prescribed. Additionally, both groups performed cadence-specific exercise compromising two interval sessions (Monday and Thursday) and two rides at constant speed (Tuesday and Friday) per week. During each interval session, cyclists completed 6 (week 1) to 8 bouts (week 4) of high intensity at 120–140 rpm and 60 rpm in HCT and LCT, respectively. The work to rest ratio of 3 min intervals was 1:1. The constant rides required the maintenance of either high (HCT) or low (LCT) pealing frequencies at submaximal intensity over 45 min. The intensity of the training was controlled by establishing heart rate targets at the equivalent percentages of the individual anaerobic threshold, which was assessed in an incremental ergometer test prior to the intervention. During the sessions, participants in both groups had to adjust the resistance manually to match the predefined heart rate targets at the prescribed cadence. The training programmes were progressive, meaning that duration of exercise at high intensity increased over the 4-week period. In both HCT and LCT, prescribed duration and intensity were identical to allow heart rate matched comparisons. Heart rate, time and cadence during sessions, including basic endurance training as well, were recorded with heart rate monitors (Polar Electro, CS 600x, Finland) coupled with a cadence sensor (Polar Electro, T1, Finland). Cadence-specific training was performed on indoor cycles (Schwinn, Johnny G 20 Spinner Pro, Canada) at an environmental temperature of 20°C.

Statistics

The statistical analysis was performed with the software SPSS 21.0 (IBM Statistics, USA) for Windows. Prior to analysis of training effects, Gaussian distribution of the data was verified by the Shapiro–Wilk Test. By using a one-way ANOVA, participants' characteristics at baseline (age, body mass, height, P_{IANS}, VO_{2MAX}, P_{MAX}), compliance to training prescriptions (heart rate, time) and delta values (pre–post) of resting EEG were compared between groups. Effects of HCT and LCT on alpha/beta ratio were analysed by applying a 2 (group: LCT, HCT) × 2 (time: pre, post) × 5 (exercise segment: W1, M1, M2, M3, CD) ANOVA. Subsequently, frequency (alpha, beta) was included as additional factor to assess the effect of HCT and LCT on EEG spectral power. As a result, main effects and interactions are reported. Furthermore, changes of EEG spectral power from warm-up were individually compared within the baseline and final assessment by using student's *T*-test for paired samples. The level of significance was set at $P \leq 0.05$.

3. Results

Apart from two female cyclists (HCT: $n = 1$; LCT: $n = 1$), who terminated the study due to personal reasons, all participants completed baseline and final exercise testing. Between HCT and LCT, there were no differences in anthropometric measurements (age: $\boldsymbol{F}(1,18) = 0.01$; $P = 0.996$; body mass: $\boldsymbol{F}(1,18) = 0.40$; $P = 0.532$; height: $\boldsymbol{F}(1,18) = 0.66$; $P = 0.427$) and aerobic power (VO_{2MAX}: $\boldsymbol{F}(1,18) = 0.97$; $P = 0.336$; P_{MAX}: $\boldsymbol{F}(1,18) = 0.87$; $P = 0.361$; P_{IANS}: $\boldsymbol{F}(1,18) = 1.22$; $P = 0.282$) prior to the intervention period. Both groups completed the required basic endurance training (HCT: 04:00:40 ± 00:08:52 h:min:s; LCT: 04:00:40 ± 00:11:13 h:min:s; $\boldsymbol{F}(1,18) = 0.01$; $P = 0.940$) at a comparable intensity (HCT: 124 ± 6 bpm; LCT: 125 ± 8 bpm; $\boldsymbol{F}(1,18) = 0.15$; $P = 0.707$). A similar exercise adherence between HCT and LCT was also given for the cadence-specific exercise, as participants in both groups attended all scheduled sessions. Furthermore, no group differences in exercise intensity were observed during interval sessions (HCT: 145 ± 5 bpm; LCT: 147 ± 4 bpm; $\boldsymbol{F}(1,18) = 0.70$; $P = 0.413$) and constant rides (HCT: 149 ± 4 bpm; 17 LCT: 148 ± 5 bpm; $\boldsymbol{F}(1,18) = 0.20$; $P = 0.660$).

Regarding EEG during exercise, there was an interaction of time and group for alpha/beta power ($\boldsymbol{F}(1,18) = 5.22$; $P = 0.035$; $eta^2 = 0.225$). Before the training intervention, alpha/beta ratio increased significantly from warm-up to exercise at P_{IANS} (Figure 1) in both LCT (M1: $T(9) = -2.55$; $P = 0.031$; M2: $T(9) = -3.90$; $P = 0.004$; M3: $T(9) = -4.52$; $P = 0.001$) and HCT (M1: $T(9) = -2.69$; $P = 0.025$; M2: $T(9) = -2.63$; $P = 0.017$; M3: $T(9) = -2.37$; $P = 0.042$). Following LCT alpha/beta ratio remained increased over warm-up at M2 ($T(9) = -2.35$; $P = 0.043$) and M3 ($T(9) = -2.64$; $P = 0.042$). In contrast, there was no significant change from warm-up to any other measuring time point after HCT.

When frequency was included as additional factor, the statistical analysis revealed an interaction of time, group and frequency ($\boldsymbol{F}(1,18) = 10.08$; $P = 0.005$; $eta^2 = 0.359$). HCT reduced relative EEG spectral power in the beta band at warm-up (from 162.9 ± 51.5 to 105.5 ± 35.2%; $T(9) = 4.95$; $P = 0.001$), M1 (from 202.2 ± 93.2 to 128.8 ± 43.6%; $T(9) = 2.93$; $P = 0.017$), M2 (from 202.9 ± 103.5 to 123.6 ± 58.8%; $T(9) = 3.90$; $P = 0.004$), M3 (from 227.3 ± 107.6 to 102.9 ± 44.4%; $T(9) = 4.79$; $P = 0.001$) and CD (from 133.6 ± 46.9 to 68.1 ± 30.1%; $T(9) = 4.95$; $P = 0.001$), whereas no change was observed in LCT. In both groups, alpha power during exercise did not change over the intervention period (Table 1). Furthermore, delta values of resting EEG alpha

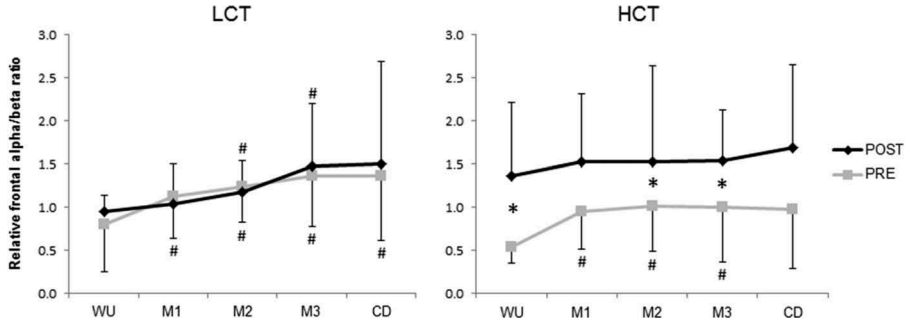

Figure 1. Change of frontal alpha/beta ratio during exercise before and after low (LCT) and high cadence training (HCT). #$P \leq 0.05$ compared to Warm-Up; *$P \leq 0.05$ compared to the same measuring time point at PRE; WU = Warm-Up (m: 100 W; f: 80 W); M1 = 10th min at individual anaerobic threshold (PIANS); M1 = 20th min at PIANS; M1 = 30th min at PIANS; CD = Cool-down (m: 100 W; f: 80 W).

Table 1. Comparison of delta value (pre–post) of EEG spectral power between low cadence (LCT; $n = 10$) and high cadence training group (HCT; $n = 10$). Values given are mean ± SD.

	Group	Rest	Warm-up	M1	M2	M3	Cool-down
ΔAlpha (pre–post)	LCT	−0.006 ± 0.017	−13.1 ± 80.1	20.1 ± 64.2	22.8 ± 71.8	29.8 ± 63.4	32.6 ± 95.8
	HCT	−0.003 ± 0.027	−37.4 ± 66.9	8.3 ± 96.5	25.3 ± 39.2	43.1 ± 73.4	13.3 ± 36.7
ΔBeta (pre–post)	LCT	−0.008 ± 0.016	13.2 ± 44.5	−1.0 ± 67.4	14.2 ± 35.2	18.1 ± 34.0	21.9 ± 36.2
	HCT	−0.006 ± 0.013	57.5 ± 36.7*,#	73.4 ± 79.3*,#	79.3 ± 64.3*,#	124.4 ± 82.2*,#	65.5 ± 41.8*,#

*$P \leq 0.05$ HCT vs LCT; #$P \leq 0.05$ pre vs post training values; M1 = 10th min at workload at individual anaerobic threshold (P_{IANS}); M2 = 20th min at P_{IANS}; M3 = 30th min at P_{IANS}

Table 2. Changes of maximal oxygen consumption (VO_{2MAX}), maximal workload (P_{MAX}) and workload at the individual anaerobic threshold (P_{IANS}) after high (HCT; $n = 10$) and low cadence training (LCT; $n = 10$). Values given are mean ± SD.

	Group	Pre	Post
VO_{2MAX} ($ml \cdot min^{-1} \cdot kg^{-1}$)	LCT	48.6 ± 4.8	52.51 ± 5.2*
	HCT	50.8 ± 5.7	55.46 ± 6.0*
P_{MAX} (W)	LCT	305.1 ± 59.4	329.3 ± 62.9*
	HCT	327.8 ± 54.5	350.5 ± 57.1*
P_{IANS} (W)	LCT	208.0 ± 44.4	231.8 ± 46.6*
	HCT	231.0 ± 52.7	260.9 ± 51.5*

*$P \leq 0.05$ pre vs. post training values

(***F***(1,18) = 0.02; $P = 0.886$) and beta power (***F***(1,18) = 3.34; $P = 0.084$) were not significantly different between groups.

Regarding aerobic performance, a main effect of time on P_{MAX} (***F***(1,18) = 49.12; $P \leq 0.001$; $eta^2 = 0.711$), P_{IANS} (***F***(1,18) = 104.02; $P \leq 0.001$; $eta^2 = 0.839$) and VO_{2MAX} (***F***(1,18) = 70.41; $P \leq 0.001$; $eta^2 = 0.779$) was observed (Table 2). However, changes were not significantly different between HCT and LCT (P_{MAX}: ***F***(1,18) = 0.05; $P = 0.825$; P_{IANS}: ***F***(1,18) = 1.337; $P = 0.261$; VO_{2MAX}: ***F***(1,18) = 0.54; $P = 0.472$).

4. Discussion

In line with the transient hypofrontality hypothesis (Dietrich, 2006), previous studies have shown that an increase in exercise intensity causes a deactivation of the frontal lobe (Brümmer et al., 2011; Schneider et al., 2009). The shift of cortical activity away from regions not involved in the demanding exercise task is considered to be due to the limited resources of the brain. In LCT, this redistribution of activity was observed from warm-up to exercise at the individual anaerobic threshold at baseline and after the intervention period. In contrast, HCT only showed a frontal deactivation at baseline assessment. After HCT, cyclists' frontal alpha/beta ratio did not change over the exercise duration. This implies that the intensity of exercise was no longer high enough to cause a redistribution of cortical activity towards regions involved in planning and executing motor commands (Dietrich, 2006). One possible explanation is a more efficient use of the brain's resources, because exercising at high pedalling frequencies allowed cyclists to complete an exercise bout of similar load with less brain cortical activity. This was shown by an increased alpha/beta ratio over the exercise duration, which was mainly due to decreased beta activity at the final assessment.

The observed functional adaptation of the brain after HCT might be interpreted in terms of the neural efficiency hypothesis, which posits that experts compared to non-experts complete a task with less neural activation particularly in the frontal region (Neubauer & Fink, 2009). Del Percio et al. (2009) have previously confirmed neural efficiency during movement by using a cross-sectional design. The authors observed that athletes compared to untrained individuals showed a lower task-related decrease in alpha power, which indicates a lower increase in cortical activation. Untrained individuals recruit additional cortical resources, which might not be essential for performing the task (Babiloni et al., 2010; Del Percio et al., 2011). In this respect, Ludyga et al. (2015b) have also shown that cyclists with higher aerobic fitness compared to peers with lower aerobic fitness complete a cycling bout with less cortical activity. In the present study, improvements of maximal oxygen uptake were accompanied by a decrease in cortical activity during exercise only after HCT. This indicates enhanced neural efficiency in HCT, which might allow a reservation of cortical resources for prolonged duration or

progressing workloads. The reduced cortical activity observed after HCT might, therefore, predict an enhanced capacity for resource allocation in a sports-specific task. Evidence from animal studies suggests that such enhancement of neural efficiency is due to improved neural processing and increasing influence of the cerebellar–thalamic–cortical circuit (Holschneider, Yang, Guo, & Maarek, 2007). Taniwaki et al. (2006) assume that this increased influence is caused by a change from internal to increasingly external and automatic processing of motor tasks. Consequently, the lower level of arousal during exercise could be explained by cycling becoming an increasingly automated process after HCT.

Furthermore, reductions of the alpha/beta ratio could be due to the increased psycho-physiological stress, which is associated with cycling at 120 rpm (Hottenrott et al., 2013). Jain, Gourab, Schindler-Ivens and Schmit (2013) have shown that a peak in cortical activity is reached, when legs are transitioned from extension to flexion and vice versa. Comparing high versus low cadence exercise, the frequency of transitions is doubled. This also requires an increased rate of afferent signalling (Jain et al., 2013) and might in turn be accompanied with more frequent sensory feedback from muscles and the cardiorespiratory system. Therefore, HCT is suggested to induce greater processing demands, which are expected to provoke a familiarisation with afferent signals (Nybo & Secher, 2004). A reduced arousal level during cycling at 90 rpm, which is reflected by a higher frontal alpha/beta ratio (Nielsen et al., 2001; Nielsen & Nybo, 2003), might be explained by an adaptation to the processing of peripheral feedback after HCT. Consequently, a lowering of cortical activity in the frontal region as described in the transient hypofrontality hypothesis (Dietrich, 2006) was not required, because a familiarisation with an increased rate of afferent signalling might have allowed a reservation of the brain's resources. This assumption is supported by Schneider et al. (2009), who have confirmed a lower level of arousal in participants performing familiar exercise compared with unfamiliar exercise.

In contrast to changes in brain cortical activity, four weeks of HCT and LCT elicited similar benefits on aerobic performance. Previous trials with a similar intervention length have shown that LCT compared to exercise at high pedalling frequencies leads to greater improvements of peak power and/or maximal oxygen consumption (Nimmerichter, Eston, Bachl, & Williams, 2012; Paton, Hopkins, & Cook, 2009). These conflicting results might be due to methodological differences as both studies included cadence-specific training programmes, which were not matched by heart rate or work. However, similar improvements in both the high and low cadence group implicates that these favourable changes are merely due to the chosen intensity and duration of the training programmes.

5. Limitations and future directions

So far, the underlying mechanisms for training-induced adaptations of brain cortical activity remain unclear. Future studies should therefore aim to combine both EEG and fMRI to gain more insights on the interaction of brain cortical activity and morphology. Furthermore, our results have to be interpreted with caution as the present study focused on changes of brain cortical activity in the frontal brain region only. Adaptations to high or LCT might as well have occurred at other electrode sites. From a practical perspective, the performance improvements observed in the present study encourage the integration of exercise sessions at low and high pedalling frequencies into the training process of cyclists. However, further benefits might be elicited by HCT as adaptations of the central nervous system were confirmed only after training at high pedalling frequencies. Although the intensity of HCT and LCT was matched by similar heart rate targets, it cannot be excluded that differences in workload between the groups have contributed to the observed exercise adaptations. At fixed workload (submaximal intensity), a higher cadence goes along with an increased heart rate (Gottshall, Bauer, & Fahrner, 1996). Conversely, cyclists exercising at a low cadence compared to participants cycling at a high cadence need to increase workload to reach similar heart rate targets. Due to this relationship between cadence, workload and heart rate, it might be assumed that LCT completed exercise training at higher workloads than HCT. Therefore, future studies should seek to investigate changes in cortical activity after training at different workloads, while cadence is held constant between the programmes.

6. Conclusion

Training at high or low pedalling frequencies elicited similar aerobic performance enhancements after four weeks. Despite having heart rate matched programmes, adaptations of brain function have been observed after HCT only. Thus, regular exercise at high pedalling frequencies enables cyclists to perform an exercise bout at the individual anaerobic threshold with less cortical activation in the frontal lobe. Additionally, high cadence trained cyclists maintain a stable level of brain cortical activity during exercise, which might allow a reservation of cortical resources for prolonged exercise.

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Disclosure statement

No potential conflict of interest was reported by the authors.

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